DIALOG
08/478,387
9-2-96
CHF 1D, 1M

8/5/1 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

125083348 CA: 125(7)83348t JOURNAL
Loss of retinoblastoma gene (RB1) is associated with deletions at the 17p13.3 chromosome and S-phase index in human breast cancer AUTHOR(S): Venesio, Tiziana; Bernardi, Amelia; Scordamaglia, Antonella; Ferrero, Paolo; Salvego, Monica; Cappa, Alberto P. M.; Liscia, Daniel S. LOCATION: Servizio di Anatomia Patologica, Dipartimento di Oncologia, 10123, Turin, Italy
JOURNAL: Ann. N. Y. Acad. Sci. DATE: 1996 VOLUME: 784 NUMBER: Basis for Cancer Management PAGES: 462-466 CODEN: ANYAA9 ISSN: 0077-8923 LANGUAGE: English
SECTION:
CA214001 Mammalian Pathological Biochemistry
CA203XXX Biochemical Genetics
IDENTIFIERS: breast cancer gene RB1 chromosome 17p13
DESCRIPTORS:
Genetic element...
D17S5; loss of retinoblastoma gene (RB1) is assocd. with deletions at 17p13.3 chromosome and S-phase index in human breast cancer Chromosome,human 17... Gene,animal, RB1... Interphase,biological, S-phase ... Mammary gland,neoplasm, carcinoma... Mutation,deletion... Ribonucleic acid formation factors,gene Rb...
loss of retinoblastoma gene (RB1) is assocd. with deletions at 17p13.3 chromosome and S-phase index in human breast cancer

8/5/2 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123248552 CA: 123(19)248552d PATENT
Hybridization probes for detection of genetic alterations that correlate with lung carcinomas
INVENTOR(AUTHOR): Christman, Michael F.; Gray, Joe W.; Levin, Nikki A.; Brzoska, Pius; Nakamura, Haruhiko
LOCATION: USA
ASSIGNEE: Regents of the University of California
PATENT: PCT International ; WO 9522624 A1 DATE: 950824
APPLICATION: WO 95US346 (950111) *US 199772 (940222)
PAGES: 39 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12Q-001/68A DESIGNATED COUNTRIES: CA; JP DESIGNATED REGIONAL: AT; BE; CH; DE; DK; ES ; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE
SECTION:
CA203001 Biochemical Genetics
CA209XXX Biochemical Methods
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: lung cancer diagnosis chromosomal aberration detection, comparative genome hybridization lung cancer diagnosis

DESCRIPTORS:
Mutation...
amplification, assocd. with lung cancer, detection of; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Nucleotides,oligo-, biotinylated,biological studies...
as hybridization probes; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Mutation,deletion...
assocd. with lung cancer, detection of; hybridization probes for detection of genetic alterations that correlate with lung carcinomas Nucleic acid hybridization...
CGH (comparative genome hybridization); hybridization probes for detection of genetic alterations that correlate with lung carcinomas Nucleotides,oligo-,biological studies...
digoxigenin-labeled, as hybridization probes; hybridization probes for detection of genetic alterations that correlate with lung carcinomas Nucleotides,oligo-,biological studies...
fluorescein-labeled, as hybridization probes; hybridization probes for detection of genetic alterations that correlate with lung carcinomas Chromosome...
human, amplification or deletion in lung cancer; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Lung,neoplasm... Lung,neoplasm, non-small-cell carcinoma... hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Lung,neoplasm, small-cell carcinoma...
small cell; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Chromosome,human X...
Xq26, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Chromosome,human 1...
1p, 1q24, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Chromosome,human 10...
10q26, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Chromosome,human 16...
16p11.2, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Chromosome,human 17...
17p, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Chromosome,human 19...
19p13.3, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas

Chromosome,human 22...
22q12.1-13.1, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas
Chromosome,human 3...
3q, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas Chromosome,human 5...
5p, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas Chromosome,human 8...
8q, detection of amplification or deletion in; hybridization probes for detection of genetic alterations that correlate with lung carcinomas

8/5/3 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

123220263 CA: 123(17)220263m PATENT
Repeat sequence chromosome-specific nucleic acid probes and methods for their preparation and use
INVENTOR(AUTHOR): Weier, Heinz Ulrich G.; Gray, Joe W.
LOCATION: USA
ASSIGNEE: Reagents of the University of California
PATENT: United States ; US 5427932 A DATE: 950627
APPLICATION: US 858124 (920326) *US 683441 (910409)
PAGES: 32 pp. Cont.-in-part of U.S. Ser. No. 683,441, abandoned. CODEN: USXXAM LANGUAGE: English CLASS: 435091200; C12P-019/34A; C07H-021/04B SECTION:
CA203001 Biochemical Genetics
CA213XXX Mammalian Biochemistry
IDENTIFIERS: human chromosome repetitive DNA hybridization probe, alphoid DNA human chromosome hybridization probe
DESCRIPTORS:
Chromosome... Chromosome,human 10... Chromosome,human 17... Chromosome,human 3... Chromosome,human 8... Deoxyribonucleic acids,satellite, .alpha.... Nucleic acid hybridization,in situ... Polymerase chain reaction...
repeat sequence chromosome-specific nucleic acid probes and methods for their prepn. and use
CAS REGISTRY NUMBERS:
145719-48-8 arbitrary PCR primer Jun1; repeat sequence chromosome-specific nucleic acid probes and methods for their prepn. and use 145895-86-9P 145895-87-0P nucleotide sequence; repeat sequence chromosome-specific nucleic acid probes and methods for their prepn. and use
168117-07-5 PCR primer WA1; repeat sequence chromosome-specific nucleic acid probes and methods for their prepn. and use
168117-09-7 PCR primer WA11; repeat sequence chromosome-specific nucleic acid probes and methods for their prepn. and use
168117-10-0 PCR primer WA12; repeat sequence chromosome-specific nucleic acid probes and methods for their prepn. and use
168117-08-6 PCR primer WA2; repeat sequence chromosome-specific

nucleic acid probes and methods for their prepn. and use

8/5/4 (Item 4 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122206754 CA: 122(17)206754v JOURNAL
Gains and losses of DNA sequences in osteosarcomas by comparative genomic hybridization.
AUTHOR(S): Tarkkanen, Maija; Karhu, Ritva; Kallioniemi, Anne; Elomaa, Inkeri; Kivioja, Aarne H.; Nevalanian, Juha; Boehling, Tom; Karaharju, Erkki; Hyytinen, Eija; et al.
LOCATION: Dep. Med. Genetics and Pathology, Univ. Helsinki, FIN-00014, Helsinki, Finland
JOURNAL: Cancer Res. DATE: 1995 VOLUME: 55 NUMBER: 6 PAGES: 1334-8 CODEN: CNREA8 ISSN: 0008-5472 LANGUAGE: English
SECTION:
CA203003 Biochemical Genetics
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: gene osteosarcoma mapping
DESCRIPTORS:
Bone,neoplasm, osteosarcoma...
genes assocd. with; identification of chromosomal regions likely to harbor previously unreported genes assocd. with human osteosarcoma Chromosome,human X... Chromosome,human 10... Chromosome,human 12... Chromosome,human 17... Chromosome,human 2... Chromosome,human 3... Chromosome,human 6... Chromosome,human 8...
identification of chromosomal regions likely to harbor previously unreported genes assocd. with human osteosarcoma Gene,animal...
osteosarcoma-assocd.; identification of chromosomal regions likely to harbor previously unreported genes assocd. with human osteosarcoma

8/5/5 (Item 5 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122184353 CA: 122(15)184353j JOURNAL
Identification of gains and losses of DNA sequences in primary bladder cancer by comparative genomic hybridization
AUTHOR(S): Kallioniemi, Anne; Kallioniemi, Olli-P.; Citro, Gil; Sauter, Guido; DeVries, Sandy; Kerschmann, Russell; Caroll, Peter; Waldman, Fred LOCATION: Department of Laboratory Medicine, Tampere University Hospital, Tampere, Finland
JOURNAL: Genes, Chromosomes Cancer DATE: 1995 VOLUME: 12 NUMBER: 3 PAGES: 213-19 CODEN: GCCAES ISSN: 1045-2257 LANGUAGE: English SECTION:
CA214001 Mammalian Pathological Biochemistry
CA203XXX Biochemical Genetics
IDENTIFIERS: gene bladder cancer comparative genomic hybridization DESCRIPTORS:

Nucleic acid hybridization...
comparative genomic; detection of DNA sequence gains and losses along human chromosomes in primary bladder cancer using comparative genomic hybridization
Chromosome,human 1... Chromosome,human 11... Chromosome,human 12... Chromosome,human 13... Chromosome,human 17... Chromosome,human 3... Chromosome,human 8... Chromosome,human 9...
detection of DNA sequence gains and losses along human chromosomes in primary bladder cancer using comparative genomic hybridization Mutation...
in human chromosomes in primary bladder cancer as identified using comparative genomic hybridization
Gene,animal... Gene,animal, anti-onco-...
in primary bladder cancer as identified using comparative genomic hybridization

8/5/6 (Item 6 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

118001986 CA: 118(1)1986t PATENT
Chromosome-specific hybridization probes for use in karyotyping
INVENTOR(AUTHOR): Gray, Joel W.; Pinkel, Daniel
LOCATION: USA
ASSIGNEE: University of California
PATENT: Canada ; CA 1301605 A1 DATE: 920526
APPLICATION: CA 526751 (870116) *US 819314 (860116)
PAGES: 55 pp. CODEN: CAXXA4 LANGUAGE: English CLASS: C12Q-001/68A; G01N-001/30B; G01N-033/52B; G01N-033/58B
SECTION:
CA203001 Biochemical Genetics
CA209XXX Biochemical Methods
IDENTIFIERS: chromosome specific unique sequence hybridization probe DESCRIPTORS:
Genetic vectors,cosmid... Plasmid and Episome...
Virus,bacterial... carrying chromosome-specific unique sequences, as hybridization probe for in situ identification of individual chromosomes
Nucleic acid hybridization,in situ...
chromosome-specific and heterogeneous unique sequence probes for Down's syndrome...
diagnosis by in situ hybridization of, heterogeneous chromosome-specific unique sequences as probes in
Chromosome,human X... Chromosome,human Y... Chromosome,human 1... Chromosome,human 10... Chromosome,human 11... Chromosome,human 12... Chromosome,human 13... Chromosome,human 14... Chromosome,human 15... Chromosome,human 16... Chromosome,human 17... Chromosome,human 18... Chromosome,human 19... Chromosome,human 2... Chromosome,human 20... Chromosome,human 21... Chromosome,human 22... Chromosome,human 3... Chromosome,human 4... Chromosome,human 5... Chromosome,human 6... Chromosome,human 7... Chromosome,human 8... Chromosome,human 9...
heterogeneous unique sequence hybridization probes specific

for, for in situ hybridization
Chromosome...
heterogeneous unique sequence hybridization probes specific for individual, for in situ hybridization
Genome,aneuploidy... Recombination,genetic, rearrangement...
identification by in situ hybridization of, heterogeneous chromosome-specific unique sequences as probes in
Molecular cloning...
of chromosome-specific unique sequences, in prepn.
hybridization probes for karyotyping
Deoxyribonucleic acid sequences,repetitive...
removal from chromosome-specific DNA banks of, in prepn. heterogeneous unique sequence probes for in situ hybridization
Deoxyribonucleic acid sequences...
unique, as hybridization probes specific for individual chromosomes in in situ hybridization

8/5/7 (Item 7 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

117186050 CA: 117(19)186050d PATENT
Chromosome-specific staining with hybridization probes to detect genetic rearrangements diagnostic of cancer
INVENTOR(AUTHOR): Gray, Joe W.; Pinkel, Daniel; Kallioniemi, Olli Pekka; Kallioniemi, Anne; Sakamoto, Masaru
LOCATION: USA
ASSIGNEE: University of California
PATENT: European Pat. Appl. ; EP 500290 A2 DATE: 920826
APPLICATION: EP 92301266 (920217) *US 659974 (910222) *US 670242 (910315) PAGES: 35 pp. CODEN: EPXXDW LANGUAGE: English
CLASS: C12Q-001/68A DESIGNATED COUNTRIES: AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LI; LU; MC; NL; PT; SE
SECTION:
CA203001 Biochemical Genetics
CA209XXX Biochemical Methods
IDENTIFIERS: chromosome staining hybridization probe cancer diagnosis DESCRIPTORS:
Genetics,cyto-, mol....
chromosome staining in, high complexity hybridization probes for, identification of genetic rearrangements diagnostic of cancer with Bone,neoplasm, osteosarcoma... Eye,neoplasm, retinoblastoma... Kidney,neoplasm, renal cell carcinoma... Lung,neoplasm... Lung,neoplasm, small-cell carcinoma... Mammary gland,neoplasm... Neoplasm... Ovary,neoplasm... Uterus,neoplasm...
detection of, by chromosome staining, high complexity hybridization probes for
Genome,aneuploidy... Recombination,genetic... Recombination,genetic, amplification... Recombination,genetic, inversion... Recombination,genetic, translocation...
detection of, by chromosome staining with high complexity

hybridization probes, cancer diagnosis in relation to
Gene,animal, RB1...
genetic rearrangement in, detection by chromosome staining
of, high complexity hybridization probes for
Nucleic acid hybridization...
in chromosome staining, with high complexity probes, for
genetic rearrangement detection and cancer diagnosis
Chromosome,human 13... Chromosome,human 17... Chromosome,human
3... rearrangements on, detection by staining of, high
complexity hybridization probes for

8/5/8 (Item 8 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

112049864 CA: 112(7)49864e TECHNICAL REPORT
Involvement of recessive oncogenes in development of
osteosarocoma AUTHOR(S): Ishizaki, Kanji
LOCATION: Radiat. Biol. Cent., Kyoto Univ., Kyoto, Japan,
JOURNAL: Kyoto Daigaku Genshiro Jikkensho, (Tech. Rep.) DATE:
1989 NUMBER: KURRI-TR-320, "Hoshasen o Riyo Shita Seimei Gensho
Kaimei e no Tenbo" Senmon Kenkyukai Hokoku PAGES: 13-17 CODEN:
KDGHDH LANGUAGE: Japanese
SECTION:
CA203003 Biochemical Genetics
CA213XXX Mammalian Biochemistry
CA214XXX Mammalian Pathological Biochemistry
IDENTIFIERS: osteosarcoma human RB gene rearrangement deletion,
human chromosome heterozygosity loss osteosarcoma, retinoblastoma
gene heterozygosity human osteosarcoma
DESCRIPTORS:
Eye,retinoblastoma,neoplasm...
gene RB for, rearrangements in, development of osteosarcoma
in relation to
Bone,osteosarcoma,neoplasm...
gene RB rearrangement and loss of chromosome heterozygosity
in, of human
Mutation,deletion...
in gene RB, in human osteosarcoma
Chromosome,human 11... Chromosome,human 12... Chromosome,human
13... Chromosome,human 15... Chromosome,human 17...
Chromosome,human 18... Chromosome,human 19... Chromosome,human
20... Chromosome,human 3... Chromosome,human 6...
loss of heterozygosity of, in osteosarcoma
Recombination,genetic, rearrangement...
of gene RB, in human osteosarcoma
Gene and Genetic element,animal, transforming...
osteosarcoma involving recessive, in human
Gene and Genetic element,animal, RB1...
rearrangement of, in human osteosarcoma

8/5/9 (Item 1 from file: 5)

DIALOG(R)File 5:BIOSIS PREVIEWS(R)

10942622 BIOSIS Number: 97142622
Chromosome analysis of nine osteosarcomas
Hoogerwerf W A; Hawkins A L; Perlman E J; Griffin C A
Johns Hopkins Oncol. Cent., Room 1-109, 600 N. Wolfe Street, Baltimore, MD 21287-8934, USA
Genes Chromosomes & Cancer 9 (2). 1994. 88-92.
Full Journal Title: Genes Chromosomes & Cancer
ISSN: 1045-2257
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 007 Ref. 092503
Although recurrent chromosome abnormalities have been identified in several histologic subtypes of sarcomas, no consistent rearrangement has yet been found in osteosarcomas. Cytogenetic analyses of nine cases of osteosarcoma are reported, including seven newly diagnosed tumors and two recurrent tumors. There were seven high-grade osteosarcomas, one periosteal osteosarcoma, and one well-differentiated sarcoma. All tumors were studied in short-term primary culture. Modal number ranged from near diploid to near triploid. Seven tumors had complex karyotypes with multiple structural abnormalities; two had only normal karyotypes. The retinoblastoma gene on chromosome 13 and the TP53 gene on chromosome 17 have been involved in osteosarcoma. Five tumors had loss of a whole copy of chromosome 13, and three of these also had a loss of a whole copy of chromosome 17. However, these losses were observed in the setting of numerous other chromosome losses. Numerous structural abnormalities were observed, many involving additions of unidentified material, unbalanced translocations, or deletions. Structural abnormalities with similar breakpoints involving 6q, 8q, 9q, and 14p were seen in two or three tumors each. When the tumors in this series were added to the 18 published cases, the pericentromeric regions of chromosomes 1, 3, and 14, and segments 6q15-21, 8q24, 9q34, 12p13, 17p13, and 19q13, were found to be involved in five or more structural rearrangements. Molecular analyses of these chromosome regions may yield genes important in the pathogenesis of osteosarcoma.
Descriptors/Keywords: RESEARCH ARTICLE; HUMAN; CYTOGENETICS; KARYOTYPE; CARCINOGENESIS
Concept Codes:
*02508 Cytology and Cytochemistry-Human
*03508 Genetics and Cytogenetics-Human
*18006 Bones, Joints, Fasciae, Connective and Adipose Tissue-Pathology *24007 Neoplasms and Neoplastic Agents-Carcinogens and Carcinogenesis 10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines Biosystematic Codes:
86215 Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

8/5/10 (Item 2 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

9296737 BIOSIS Number: 43041737
DETECTION OF STRUCTURAL CHROMOSOME ABERRATIONS BY MULTICOLOR FLUORESCENCE IN-SITU HYBRIDIZATION FISH WITH GENE-SPECIFIC AND LOCUS-SPECIFIC PROBES IN OVARIAN CANCER
SAKAMOTO M; KALLIONIEMI A; KALLIONIEMI O; MATSUMURA K; YAMAKAWA K; NAKAMURA Y; YANG-FENG T; WALDMAN F; PINKEL D; GRAY J
DIV. MOL. CYTOMETRY, UCSF, CALIF. 94143.
83RD ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR CANCER RESEARCH, SAN DIEGO, CALIFORNIA, USA, MAY 20-23, 1992. PROC AM ASSOC CANCER RES ANNU MEET 33 (0). 1992. 40. CODEN: PAMRE
Language: ENGLISH
Document Type: CONFERENCE PAPER
Descriptors/Keywords: ABSTRACT HUMAN CHROMOSOME 3 17 DIAGNOSTIC METHOD ANALYTICAL METHOD
Concept Codes:
*03508 Genetics and Cytogenetics-Human
*06504 Radiation-Radiation and Isotope Techniques
*10052 Biochemical Methods-Nucleic Acids, Purines and Pyrimidines *10506 Biophysics-Molecular Properties and Macromolecules *12504 Pathology, General and Miscellaneous-Diagnostic *16501 Reproductive System-General; Methods
*16506 Reproductive System-Pathology
*24001 Neoplasms and Neoplastic Agents-Diagnostic Methods
*24004 Neoplasms and Neoplastic Agents-Pathology; Clinical Aspects; Systemic Effects
*24006 Neoplasms and Neoplastic Agents-Biochemistry
00520 General Biology-Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals
10062 Biochemical Studies-Nucleic Acids, Purines and Pyrimidines 10504 Biophysics-General Biophysical Techniques
Biosystematic Codes:
86215 Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

8/5/11 (Item 3 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

9034142 BIOSIS Number: 93019142
ACCUMULATION OF GENETIC ALTERATIONS AND PROGRESSION OF PRIMARY BREAST CANCER
SATO T; AKIYAMA F; SAKAMOTO G; KASUMI F; NAKAMURA Y
DEP. BIOCHEM., CANCER INST., 1-37-1, KAMI-IKEBUKURO, TOSHIMA-KU, TOKYO 170, JAPAN.
CANCER RES 51 (21). 1991. 5794-5799. CODEN: CNREA
Full Journal Title: Cancer Research
Language: ENGLISH
In order to detect common regions of deletion, 219 breast

tumors were examined for loss of heterozygosity at several loci on chromosomes 3p, 16q, and 17 by restriction fragment length polymorphism analysis. Allelic deletions of loci on chromosomes 3p, 13q, 16q, and 17, and amplification of the erbB2 oncogene, were analyzed and compared with histopathological and clinical features. Common regions of deletion were detected within chromosomal bands (3p13-14.3, 16q22-23, 17p13 (two separated loci), and 17q21. Concordant losses of alleles on chromosomes 3p, 13q, 16q, 17p, and 17q were observed. A significant association was detected between loss of heterozygosity on chromosomes 17p and 17q and amplification of the erbB2 oncogene (17p, P = 0.000721, by Fisher's exact test; 17q, P < 0.001, .chi.2 = 12.135). Furthermore, tumors showing highly malignant phenotypes had accumulated more genetic changes at the loci studied than those having less malignant phenotypes on the basis of histopathological classification, lymph node metastasis, and tumor size. These results suggested that accumulation of genetic alterations, including loss of function of tumor suppressor genes on chromosomes 3p, 13q, 16q, and 17, and amplification of the erbB2 oncogene, may contribute to tumor development and/or progression in primary breast cancer.
Descriptors/Keywords: HUMAN TUMOR SUPPRESSION GENE ONCOGENE ALLELES RESTRICTION FRAGMENT LENGTH POLYMORPHISM LYMPH NODE METASTASIS Concept Codes:
*03508 Genetics and Cytogenetics-Human
*15008 Blood, Blood-Forming Organs and Body Fluids-Lymphatic Tissue and Reticuloendothelial System
*16506 Reproductive System-Pathology
*24004 Neoplasms and Neoplastic Agents-Pathology; Clinical Aspects; Systemic Effects
Biosystematic Codes:
86215 Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

8/5/12 (Item 4 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

8589674 BIOSIS Number: 92054674
COMPLETE ASSOCIATION OF LOSS OF HETEROZYGOSITY OF CHROMOSOMES 13 AND 17 IN OSTEOSARCOMA
SCHEFFER H; KRUIZE Y C M; OSINGA J; KUIKEN G; OOSTERHUIS J W; LEEUW J A; KOOPS H S; BUYS C H C M
DEP. MED. GENETICS, UNIV. GRONINGEN, ANTONIUS DEUSINGLAAN 4, NL-9713 AW GRONINGEN, NETHERLANDS.
CANCER GENET CYTOGENET 53 (1). 1991. 45-56. CODEN: CGCYD
Full Journal Title: Cancer Genetics and Cytogenetics
Language: ENGLISH
Mutations in the retinoblastoma (RB1) gene are not confined to retinoblastoma, but are also involved in the development of osteosarcoma. Structural aberrations within the RB1 gene have been studied in fresh samples of eleven cases of osteosarcoma. In five cases a rearrangement was detected, one

of which was best explained as a partial duplication. The chromosomal mechanisms by which the nonmutated RB1 allele was lost appeared to be similar in frequency to those that have been reported for retinoblastoma. Loss of heterozygosity was observed for chromosomes 3, 11, 13, 17, and 22. However, when no loss of heterozygosity of chromosome 13 was detected, the other chromosomes retained their heterozygosity as well. A complete association of loss of heterozygosity of chromosomes 13 and 17 was observed. This can be taken as an indication of the involvement of another tumor suppressor gene at chromosome 17 in the initiation of osteosarcoma.
Descriptors/Keywords: HUMAN RETINOBLASTOMA RB1 GENE TUMOR SUPPRESSOR GENE CANCER INITIATION
Concept Codes:
*02508 Cytology and Cytochemistry-Human
*03508 Genetics and Cytogenetics-Human
*12503 Pathology, General and Miscellaneous-Comparative (1970-) *18006 Bones, Joints, Fasciae, Connective and Adipose Tissue-Pathology *20006 Sense Organs, Associated Structures and Functions-Pathology *24007 Neoplasms and Neoplastic Agents-Carcinogens and Carcinogenesis 11108 Anatomy and Histology, General and Comparative-Microscopic and Ultramicroscopic Anatomy
25552 Developmental Biology-Embryology-Descriptive Teratology and Teratogenesis
Biosystematic Codes:
86215 Hominidae
Super Taxa:
Animals; Chordates; Vertebrates; Mammals; Primates; Humans

8/5/13 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09469364 95399364
Identification of novel regions of altered DNA copy number in small cell lung tumors.
Levin NA; Brzoska PM; Warnock ML; Gray JW; Christman MF
Department of Radiation Oncology, University of California, San Francisco 94143, USA.
Genes Chromosomes Cancer (UNITED STATES) Jul 1995, 13 (3) p175-85, ISSN 1045-2257 Journal Code: AYV
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9512
Subfile: INDEX MEDICUS
Identification of the genetic alterations that occur in tumors is an important approach to understanding tumorigenesis. We have used comparative genomic hybridization (CGH), a novel molecular cytogenetic method, to identify the gross DNA copy number changes that commonly occur in small cell lung cancer (SCLC). We analyzed ten SCLC tumors (seven primary tumors and three metastases) from eight patients. We found frequent increases in DNA copy number on chromosome arms 5p, 8q,

3q, and Xq and frequent decreases in copy number on chromosome arms 3p, 17p, 5q, 8p, 13q, and 4p. The increase in copy number at 8q24 (MYC) and decreases at 17p13 (TP53), 13q14 (RB), and 3p have previously been identified in SCLC with other methods. Many of the other regions in which we detected common copy number changes have not been reported to be regions of common alteration in SCLC tumors. Comparison of copy number changes between a primary tumor and a metastasis from the same patient showed that they were more closely related to each other than to any of the other tumors. The results of direct CGH analysis of SCLC tumors reported here confirm the existence of copy number changes that we identified previously by using cell lines.
Tags: Female; Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.
Descriptors: *Carcinoma, Small Cell--Genetics--GE; *Chromosome Aberrations; *Lung Neoplasms--Genetics--GE; Aged; Chromosome Mapping; Chromosomes, Human, Pair 13; Chromosomes, Human, Pair 17; Chromosomes, Human, Pair 18; Chromosomes, Human, Pair 22; Chromosomes, Human, Pair 3; Chromosomes, Human, Pair 5; Chromosomes, Human, Pair 8; DNA, Neoplasm --Analysis--AN; Middle Age; X Chromosome
CAS Registry No.: 0 (DNA, Neoplasm)

8/5/14 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09077359 95007359
Genetic studies of 457 breast cancers. Clinicopathologic parameters compared with genetic alterations [see comments]
Harada Y; Katagiri T; Ito I; Akiyama F; Sakamoto G; Kasumi F; Nakamura Y; Emi M
Department of Biochemistry, Cancer Institute, Tokyo, Japan.
Cancer (UNITED STATES) Oct 15 1994, 74 (8) p2281-6, ISSN 0008-543X Journal Code: CLZ
Comment in Cancer 1994 Oct 15;74(8):2215-7
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9501
Subfile: AIM; INDEX MEDICUS
BACKGROUND. Human breast cancers frequently show loss of heterozygosity (LOH) and/or amplification at specific chromosomal regions. METHODS. To investigate the roles of these genetic alterations during tumor development and/or progression, 457 cases of primary breast cancer were examined for LOH at chromosomal regions 16q24, 17p13.3, and 17q21, and for amplification of the erb-B2 locus at 17q11.2 and the c-myc locus at 8q24. The genetic changes then were compared with lymph node metastasis, histologic type, and tumor stage. RESULTS. The LOH at 17q21 was observed more frequently in tumors of the solid, tubular type (41 of 75 [55%]) than in other types (48 of 187 [26%]) ($P < 0.0001$). The LOH at 17p13.3 was more frequent in scirrhous and solid, tubular tumors (77 of 141

[55%] and 48 of 88 [55%]) than in other types (29 of 89 [33%]) (P = 0.0004). Generally, mutations were seen more often in tumors with axillary lymph node metastases, undifferentiated tumors, and large or invasive tumors than in tumors considered less aggressive histopathologically. However, 22 tumors bearing three or more genetic alterations were found among 187 tumors histologically diagnosed as free of axillary lymph node metastasis; similarly, 12 of 122 t1 classification tumors and 4 of 89 histologically well differentiated tumors each contained three or more genetic alterations. Although these tumors would be regarded as having a relatively good prognosis on the basis of conventional clinicopathologic diagnosis, the authors suspect that, in fact, they do not. CONCLUSIONS. Patients whose tumors contain multiple genetic alterations should be treated as a new high risk group with respect to operative and/or postoperative management. Tags: Female; Human; Support, Non-U.S. Gov't

Descriptors: *Breast Neoplasms--Genetics--GE; *Chromosome Aberrations; *Chromosomes, Human, Pair 16; *Chromosomes, Human, Pair 17; *Chromosomes, Human, Pair 8; *DNA, Neoplasm--Analysis--AN; Blotting, Southern; Breast Neoplasms--Mortality--MO; Breast Neoplasms--Pathology--PA; DNA--Analysis --AN; Gene Amplification; Genes, erbB-2; Genes, myc; Genetic Markers; Heterozygote Detection; Lymphatic Metastasis; Neoplasm Staging; Polymorphism, Restriction Fragment Length; Prospective Studies CAS Registry No.: 0 (DNA, Neoplasm); 0 (Genetic Markers); 9007-49-2 (DNA)

Gene Symbol: erb-B2; c-myc

8/5/15 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08911973 94226973

Allelotype of uterine cancer by analysis of RFLP and microsatellite polymorphisms: frequent loss of heterozygosity on chromosome arms 3p, 9q, 10q, and 17p.

Jones MH; Koi S; Fujimoto I; Hasumi K; Kato K; Nakamura Y
Department of Biochemistry, Cancer Institute, Tokyo, Japan.
Genes Chromosomes Cancer (UNITED STATES) Feb 1994, 9 (2) p119-23, ISSN 1045-2257 Journal Code: AYV

Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9408
Subfile: INDEX MEDICUS

Cancers in which mutations have been identified in putative tumor suppressor genes, such as the TP53 gene, the retinoblastoma (RBI) gene, the adenomatous polyposis coli (APC) gene, and the Wilms tumor (WTI) gene, frequently show loss of the corresponding allele on the homologous chromosome. To identify locations of tumor suppressor genes involved in uterine cancer, we examined loss of heterozygosity (LOH) by using genomic probes detecting RFLPs in 35 uterine

cancers at 29 loci throughout the genome, and with highly informative microsatellite markers in 21 uterine cancers at nine putative or known tumor suppressor gene loci. High frequencies of allelic loss found at loci on 3p (71%), 9q (38%), 10q (35%), and 17p (35%) suggest that tumor suppressor genes involved in uterine carcinogenesis exist in these regions. There were no significant differences in frequencies of LOH between cancers of the uterine cervix and cancers of the uterine endometrium at any of the loci tested. Tags: Female; Human

Descriptors: *Adenocarcinoma--Genetics--GE; *Alleles; *Carcinoma, Squamous Cell--Genetics--GE; *Cervix Neoplasms--Genetics--GE; *Chromosome Aberrations; *Endometrial Neoplasms--Genetics--GE; *Polymorphism (Genetics) ; *Repetitive Sequences, Nucleic Acid; *Sequence Deletion; *Uterine Neoplasms--Genetics--GE; Adenocarcinoma--Pathology--PA; Base Sequence; Carcinoma, Squamous Cell--Pathology--PA; Cervix Neoplasms--Pathology--PA; Chromosomes, Human, Pair 10--Ultrastructure--UL; Chromosomes, Human, Pair 17--Ultrastructure--UL; Chromosomes, Human, Pair 3--Ultrastructure--UL; Chromosomes, Human, Pair 9--Ultrastructure--UL; DNA, Neoplasm--Genetics--GE ; DNA, Satellite--Genetics--GE; Endometrial Neoplasms--Pathology--PA; Genes, Suppressor, Tumor; Heterozygote; Lymphatic Metastasis--Genetics--GE; Polymorphism, Restriction Fragment Length

CAS Registry No.: 0 (DNA, Neoplasm); 0 (DNA, Satellite)
Gene Symbol: TP53; RB1; APC; WT1

8/5/16 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08671903 93381903

Tumor suppressor gene allelic loss in human renal cancers.

Brooks JD; Bova GS; Marshall FF; Isaacs WB

Department of Urology, Johns Hopkins University School of Medicine, Baltimore, Maryland.

J Urol (UNITED STATES) Oct 1993, 150 (4) p1278-83, ISSN 0022-5347 Journal Code: KC7

Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9312
Subfile: AIM; INDEX MEDICUS

It is now apparent that multiple genetic alterations, including oncogene activation and tumor suppressor gene inactivation, are necessary steps in carcinogenesis. We have studied this concept in renal cancers by looking at specific tumor suppressor genes implicated in several allelotyping studies. Primary, predominantly low stage renal tumors of varying grades and histologic subtypes were investigated for allelic loss of 3p, 17p and the p53 gene, the DCC gene and the Rb gene and its product. 3p loss occurred in 47% of tumors studied

and was much more common in clear cell cancers (85%). 17p and p53 gene loss were relatively uncommon events with only 6 of 42 tumors demonstrating loss. None of the tumors with typical histologies had allelic loss of the DCC gene, though loss did occur in leiomyosarcoma and a collecting duct tumor. Allelic loss of the Rb gene occurred in one clear cell tumor, the leiomyosarcoma, and, interestingly, in both collecting duct tumors in this series. Allelic loss of the Rb gene was correlated with little or no RB protein expression as judged by immunohistochemistry. At all loci studied, allelic loss did not appear to correlate with tumor grade or stage. These results suggest that inactivation of the p53, Rb, and DCC genes by allelic loss are uncommon events in the early stages of renal carcinogenesis.

Tags: Human

Descriptors: *Chromosome Deletion; *Chromosomes, Human, Pair 17; *Chromosomes, Human, Pair 3; *DNA, Neoplasm--Analysis--AN; *Gene Expression Regulation, Neoplastic; *Genes, Suppressor, Tumor--Genetics--GE; *Kidney Neoplasms--Genetics--GE; Blotting, Southern; Genes, p53--Genetics--GE; Genes, DCC--Genetics--GE; Genes, Retinoblastoma--Genetics--GE; Polymerase Chain Reaction

CAS Registry No.: 0 (DNA, Neoplasm)

Gene Symbol: p53; Rb; DCC

8/5/17 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08248443 92386443

Cytogenetics of epithelial malignant lesions.

Pathak S

Department of Cell Biology, University of Texas M. D. Anderson Cancer Center, Houston.

Cancer (UNITED STATES) Sep 15 1992, 70 (6 Suppl) p1660-70, ISSN 0008-543X Journal Code: CLZ

Contract/Grant No.: RR04999, RR, NCRR

Languages: ENGLISH

Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL

JOURNAL ANNOUNCEMENT: 9212

Subfile: AIM; INDEX MEDICUS

Chromosomal abnormalities have been believed to be responsible for neoplastic transformation and tumor growth for a long time. The confirming observations are of two types: (1) primary cytogenetic alterations that are responsible for tumor initiation and (2) secondary abnormalities that are acquired late and are associated with tumor growth, heterogeneity, and metastasis. Primary chromosomal abnormalities (such as the 13q deletion in retinoblastoma, 11p deletion in Wilms' tumor, 3p anomalies in renal cell carcinoma, and 5q deletion in colorectal carcinomas) first were identified in lymphocyte cultures as constitutional defects. Later, similar types of defects were observed as tumor-specific aberrations from patients whose

lymphocytes otherwise had normal chromosomes. Recently, it has become clear that classes of known cancer-related genes (dominant protooncogenes and recessive tumor-suppressor or anti-oncogenes) are located at those hot spots that are involved in neoplasia-associated chromosomal alterations. In breast carcinoma, such a specific chromosomal alteration has not been identified conclusively in lymphocyte cultures, although chromosome 1 alterations have been observed in cell lines, directly processed effusions, and primary breast tumors. Lymphocyte cultures; primary tumors; and established cell lines from breast carcinomas, colorectal carcinomas, and renal cell carcinomas were analyzed to identify (1) primary chromosomal alterations precisely and (2) secondary cytogenetic defects that are associated with these most common solid adult neoplasms. Peripheral blood analysis indicated that chromosomes 1, 17, and 18 in breast carcinomas; chromosomes 3 and 14 in renal cell carcinomas; and chromosomes 5, 12, and 17 in colorectal carcinomas were involved nonrandomly in structural anomalies in a small number of lymphocyte cells (1-4%). These chromosomal aberrations were considered primary defects because of their involvement as marker formations in tumor cells; other structural and numeric abnormalities also were found. These results indicate that lymphocyte chromosomal analysis might identify those at high risk for breast, colorectal, and renal cell carcinomas, among other malignant lesions. Such identifications could facilitate early selection for primary and secondary cancer prevention or interventional trials. (73 Refs.)

Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S. Descriptors: *Chromosome Aberrations; *Chromosome Abnormalities--Genetics --GE; *Neoplasms--Genetics--GE; Genes, Suppressor, Tumor; Oncogenes

8/5/18 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07959051 92097051

Second relapse of acute promyelocytic leukemia (ANLL-M3) with t(15;17) and t(1;3)(p36;q21).

Sato Y; Murai M; Tsunoda J; Komatsu N; Muroi K; Yoshida M; Motoyoshi K; Sakamoto S; Miura Y

Department of Medicine, Jichi Medical School, Tochigi-ken, Japan. Cancer Genet Cytogenet (UNITED STATES) Nov 1991, 57 (1) p53-8, ISSN 0165-4608 Journal Code: CMT

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9204

Subfile: INDEX MEDICUS

We describe herein a patient with acute promyelocytic leukemia (APL)-(ANLL-M3) whose bone marrow cells in the second relapse showed t(1;3)(p36;q21) together with t(15;17)(q22;q11-q12). Although a total of 21 patients with t(1;3) have been reported so far, among which three cases with de novo

acute nonlymphocytic leukemia were included, our patient is the first case with APL. The hematologic findings in our case confirmed the previous observations that this anomaly is associated with relatively high platelet count and the multi-myeloid lineage involvement of leukemic cells. Our patient responded well to chemotherapy and achieved first and second remission with 42 months of total survival, contrary to our expectation that patients with this anomaly have a poor prognosis.

Tags: Case Report; Human; Male; Support, Non-U.S. Gov't

Descriptors: *Leukemia, Promyelocytic, Acute--Genetics--GE; Chromosomes, Human, Pair 1; Chromosomes, Human, Pair 15; Chromosomes, Human, Pair 17; Chromosomes, Human, Pair 3; Leukemia, Promyelocytic, Acute--Pathology--PA; Leukemia, Promyelocytic, Acute--Therapy--TH; Middle Age; Recurrence; Translocation (Genetics)

8/5/19 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07867465 92005465

Molecular genetic alterations in superficial and locally advanced human bladder cancer.

Presti JC Jr; Reuter VE; Galan T; Fair WR; Cordon-Cardo C
Department of Surgery, Memorial Sloan-Kettering Cancer Center, New York, New York 10021.

Cancer Res (UNITED STATES) Oct 1 1991, 51 (19) p5405-9, ISSN 0008-5472 Journal Code: CNF

Contract/Grant No.: CA47538, CA, NCI

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 9201

Subfile: INDEX MEDICUS

We attempted to define the role of tumor suppressor genes in the pathogenesis of human bladder cancer through a combined molecular genetic and immunohistochemical approach. Thirty-four bladder tumors (1 Pis, 6 Pa, 5 P1, 3 P3a, 18 P3b, 1 P4; 8 low grade and 26 high grade tumors) have been analyzed. Restriction fragment length polymorphism analysis directed at 5 suspected or established tumor suppressor gene regions (3p21-25, 11p15, 13q14, 17p11-13, and 18q21) was combined with immunohistochemical using Rb-PMG3-245 monoclonal antibody directed at the retinoblastoma (Rb) gene product. Tumor grade correlated with deletions of 3p ($P = 0.004$) and 17p ($P = 0.063$). Tumor stage correlated with deletions of 3p ($P = 0.010$), 17p ($P = 0.015$) and altered Rb expression ($P = 0.054$). Vascular invasion correlated only with deletions of 17p ($P = 0.038$). No marker correlated with positive lymph nodes. Our results suggest that altered Rb expression occurs in all grades and stages of bladder cancer but is more commonly associated with invasive tumors. Genetic alterations of 3p, 11p, 17p, and 18q are rare events in low grade, superficial tumors, whereas they are more

common in high grade and invasive bladder cancer. The role of these genetic alterations in the prognosis of bladder cancer will require additional follow-up and further studies.
Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S. Descriptors: *Bladder Neoplasms--Genetics--GE; Bladder Neoplasms --Metabolism--ME; Blotting, Southern; Chromosome Deletion; Chromosomes, Human, Pair 11; Chromosomes, Human, Pair 13; Chromosomes, Human, Pair 17; Chromosomes, Human, Pair 18; Chromosomes, Human, Pair 3; DNA--Analysis--AN; Genes, Suppressor, Tumor; Immunohistochemistry; Neoplasm Invasiveness; Neoplasm Staging; Polymorphism, Restriction Fragment Length; Retinoblastoma Protein--Biosynthesis--BI
CAS Registry No.: 0 (Retinoblastoma Protein); 9007-49-2 (DNA)

8/5/20 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07843361 91362361
Chromosome anomalies in human breast cancer: evidence for specific involvement of 1q region in lymphocyte cultures.
Pathak S; Hopwood VL; Hortobagyi GN; Jackson GL; Hughes JI; Melillo D Department of Cell Biology, University of Texas M.D. Anderson Cancer Center, Houston 77030.
Anticancer Res (GREECE) May-Jun 1991, 11 (3) p1055-60, ISSN 0250-7005 Journal Code: 59L
Contract/Grant No.: RR04999-01, RR, NCRR
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9112
Subfile: INDEX MEDICUS
Constitutional chromosome abnormalities that may predispose a group of individuals to develop certain neoplasms have been reported for some human solid tumors. Deletions of 13q in retinoblastoma, 11p in Wilms' tumor, 1p in neuroblastoma, 3p in renal cell carcinoma, 5q in colorectal carcinoma and 22q in meningioma are examples of such anomalies. In breast carcinoma, a specific cytogenetic defect has not been conclusively identified. We have studied Phytohemagglutinin-stimulated lymphocytes of 76 breast cancer patients, 68 predisposed family members, 40 controls, and 30 additional controls with lung cancer to determine whether nonrandom chromosome defects are present. From each sample 100, G-or Q-banded metaphase spreads were studied for rearrangements. A marked clustering of alterations in the long arm of chromosome no. 1 (q11-22) was seen in breast cancer patients and in some predisposed family members. Alterations in 1q were present in 1% to 3% of metaphases, and included translocations to chromosomes 3, 6, 7, 9, 12, 15, 17, 18, 21 and the X; deletion of 1q, or pericentric inversion. Twelve out of 62 (19.3%) familial cases, 3 out of 14 (21.4%) sporadic cases, 9 out of 68 (13.2%) predisposed cases and 2 out of 40 (5%) control cases showed 1q alterations. None of the 30 lung cancer patients showed chromosome 1 anomaly in this

region. This is consistent with the reports on primary breast tumor tissues, cell lines and pleural effusions where 1q defects have been reported. We conclude that chromosome 1q rearrangement might be one of the primary lesions specifically associated with the development of breast cancer.
Tags: Female; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S. Descriptors: *Breast Neoplasms--Genetics--GE; *Chromosome Aberrations; *Chromosomes, Human, Pair 1; *Lymphocytes--Ultrastructure--UL; Cells, Cultured; Chromosomes, Human, Pair 17; Gene Rearrangement

8/5/21 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07651166 91170166
Multiple genetic alterations in small-cell lung carcinoma.
Yokota J; Mori N; Akiyama T; Shimosato Y; Sugimura T; Terada M
National Cancer Center Research Institute, Tokyo, Japan.
Princess Takamatsu Symp (UNITED STATES) 1989, 20 p43-8,
Journal Code: HHI
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 9106
Subfile: INDEX MEDICUS
By restriction fragment length polymorphism (RFLP) analysis, it was found that loss of heterozygosity (LOH) at three different chromosomal loci, 3p, 13q, and 17p, occurs simultaneously in nearly 100% of small-cell lung carcinomas (SCLC). This was observed even in stage I tumors and an untreated tumor, and it occurred prior to NMYC amplification. The common region of LOH on chromosome 3p was 3p14-24.1, and this region was also frequently lost in carcinoma of the uterine cervix (100% at D3S2 on 3p14-21) as well as renal cell carcinoma (56% at ERBA beta on 3p22-24.1), suggesting the presence of tumor suppressor gene(s) for these cancers in this region. On chromosome 13, LOH was observed commonly in the region between 13q12 and 13q22, including the RB locus on 13q14, and normal RB protein was not detected in any of 9 SCLC cell lines by immunoprecipitation analysis. The common region of LOH on chromosome 17 was 17p13 and is the same as that in colon carcinoma and osteogenic sarcoma. Since LOH is supposed to unmask the recessive mutation of tumor suppressor gene in the remaining allele, these results may imply that at least six genetic alterations are necessary to convert a normal cell into a fully malignant cancer cell in SCLC. RFLP analysis was performed on several other types of human cancers, including carcinoma of the uterine cervix, neuroblastoma, hepatocellular carcinoma, pheochromocytoma, and stomach cancer to determine the chromosomal loci of putative tumor suppressor genes in each tumor. Chromosomal loci showing frequent LOH were different among these tumors.(ABSTRACT TRUNCATED AT 250 WORDS)
Tags: Comparative Study; Human

Descriptors: *Carcinoma, Small Cell--Genetics--GE; *Lung Neoplasms --Genetics--GE; *Polymorphism, Restriction Fragment Length; Cell Transformation, Neoplastic--Genetics--GE; Chromosomes, Human, Pair 13; Chromosomes, Human, Pair 17; Chromosomes, Human, Pair 3; Genes, Retinoblastoma; Heterozygote; Mutation; Neoplasms--Genetics--GE; Oncogenes

8/5/22 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07128659 90035659
Transcription factors and recessive oncogenes in the pathogenesis of human lung cancer.
Minna JD; Schutte J; Viallet J; Thomas F; Kaye FJ; Takahashi T; Nau M; Whang-Peng J; Birrer M; Gazdar AF
NCI-Navy Medical Oncology Branch, National Cancer Institute, Bethesda, MD 20814.
Int J Cancer Suppl (UNITED STATES) 1989, 4 p32-4, ISSN 0020-7136 Journal Code: GRM
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
JOURNAL ANNOUNCEMENT: 9002
Subfile: INDEX MEDICUS
(41 Refs.)
Tags: Human
Descriptors: *DNA-Binding Proteins--Genetics--GE; *Gene Expression Regulation, Neoplastic; *Lung Neoplasms--Genetics--GE; *Oncogenes; *Transcription Factors--Genetics--GE; Chromosomes, Human, Pair 13; Chromosomes, Human, Pair 17; Chromosomes, Human, Pair 3; Heterozygote; Oncogene Proteins--Genetics--GE; Phosphoproteins--Genetics--GE; Retinoblast oma--Genetics--GE
CAS Registry No.: 0 (DNA-Binding Proteins); 0 (Oncogene Proteins); 0 (Phosphoproteins); 0 (Protein p53); 0 (Proto-Oncogene Proteins c-jun); 0 (Transcription Factors)

8/5/23 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06531038 88176038
Genetic alterations of the c-erbB-2 oncogene occur frequently in tubular adenocarcinoma of the stomach and are often accompanied by amplification of the v-erbA homologue.
Yokota J; Yamamoto T; Miyajima N; Toyoshima K; Nomura N; Sakamoto H; Yoshida T; Terada M; Sugimura T
National Cancer Center Research Institute, Tokyo, Japan.
Oncogene (ENGLAND) Mar 1988, 2 (3) p283-7, ISSN 0950-9232 Journal Code: ONC
Languages: ENGLISH
Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 8807
Subfile: INDEX MEDICUS
We analyzed for alterations of the c-erbB-2 oncogene in 35 human stomach cancers and 8 cell lines derived from human stomach cancer. Amplification of c-erbB-2 was found in approximately 40% (5/13) of the tubular adenocarcinomas of the stomach examined, including 4 of 10 fresh tumors and one of 3 cell lines, but not in other histological types of stomach cancer examined (0/30), including 25 fresh tumors and 5 cell lines. This result strongly suggests that amplification of c-erbB-2 occurs frequently in tubular carcinomas in stomach cancer. Rearrangement of c-erbB-2 was also detected in one tubular adenocarcinoma. The rearranged fragment carried the 3' half, but not the 5' sequence, of the c-erbB-2 gene. Furthermore, one of the cellular homologues of v-erbA was amplified in 3 of 4 fresh tumors carrying the amplified c-erbB-2 gene. Both c-erbB-2 and the v-erbA homologue were expressed in all the stomach cancer cell lines tested. Tags: Human; Support, Non-U.S. Gov't
Descriptors: *Adenocarcinoma--Genetics--GE; *Oncogenes; *Proto-Oncogene Proteins--Genetics--GE; *Stomach Neoplasms--Genetics--GE; Cell Line; Chromosomes, Human, Pair 17; Chromosomes, Human, Pair 3; DNA, Neoplasm --Genetics--GE; Gene Amplification; RNA, Messenger--Genetics--GE; RNA, Neoplasm--Genetics--GE
CAS Registry No.: 0 (DNA, Neoplasm); 0 (Proto-Oncogene Proteins); 0 (RNA, Messenger); 0 (RNA, Neoplasm)

8/5/24 (Item 12 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

04768668 83001668
Multiple karyotypic changes in retinoblastoma tumor cells: presence of normal chromosome No. 13 in most tumors.
Gardner HA; Gallie BL; Knight LA; Phillips RA
Cancer Genet Cytogenet (UNITED STATES) Jul 1982, 6 (3) p201-11, ISSN 0165-4608 Journal Code: CMT
Languages: ENGLISH
Document type: JOURNAL ARTICLE
JOURNAL ANNOUNCEMENT: 8301
Subfile: INDEX MEDICUS
There are conflicting reports on the frequency in retinoblastoma tumor cells of aberrations involving chromosome No. 13. To quantitate the frequency of various chromosome aberrations, we analyzed the karyotypes from the retinoblastoma tumors; all tumors contained chromosome abnormalities. Chromosome No. 13 was altered in only two tumors, but the aberrations in these two cases affected different portions of the chromosome. We have concluded that chromosome aberrations affecting chromosome No. 13 are relatively infrequent in retinoblastoma tumors. Chromosome No.1 was involved in rearrangements in eight tumors; in six

tumors the rearrangements lead to trisomy of 1q25-1q32. Seven tumors had aberrations resulting in trisomy of the long arm of chromosome No. 17; the most common aberration was an i(17q) chromosome. Every tumor showed trisomy of the long arm of either chromosome No. 1 or 17. These changes in chromosomes No. 1 and 17 have been observed by others in many different tumors and are not unique to retinoblastoma. In summary, chromosome abnormalities were present in all retinoblastoma tumors studied, but no aberration common to all tumors was found.

Tags: Animal; Human; Support, Non-U.S. Gov't

Descriptors: *Chromosome Aberrations; *Chromosomes, Human, 13-15; *Eye Neoplasms--Genetics--GE; *Retinoblastoma--Genetics--GE; Child, Preschool; Chromosomes, Human, 1-3; Chromosomes, Human, 16-18; Infant; Inversion (Genetics); Karyotyping; Mice; Mice, Nude; Neoplasms, Experimental --Genetics--GE; Tissue Culture; Trisomy

8/5/25 (Item 13 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

04482339 82025339

A deleted chromosome no. 13 in human retinoblastoma cells: relevance to tumorigenesis.

Balaban-Malenbaum G; Gilbert F; Nichols WW; Hill R; Shields J; Meadows AT Cancer Genet Cytogenet (UNITED STATES) Apr 1981, 3 (3) p243-50, ISSN 0165-4608 Journal Code: CMT

Contract/Grant No.: CA 144896

Languages: ENGLISH

Document type: JOURNAL ARTICLE

JOURNAL ANNOUNCEMENT: 8202

Subfile: INDEX MEDICUS

In this report of banded karyotypes prepared after short-term culture (72 hr) from human retinoblastoma tumor tissue, on del(13)(pter leads to q14:) chromosome and one normal chromosome #13 were found in all of the metaphases examined. Similar deletions (always involving 13q14) have previously been described in the somatic cells of individuals with one form of retinoblastoma. In the present case, however, the constitutional karyotype is normal. The presence of tumors in both eyes suggests that this is the genetic form of retinoblastoma, even though the patient's family history is negative for this tumor. The normal constitutional karyotype argues that the chromosome deletion occurred as a postzygotic event. The modal chromosome number of the tumor cells is 47 and rearrangements involving chromosomes #2, #17, and #20 were also identified. Tags: Case Report; Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Descriptors: *Chromosome Deletion; *Chromosomes, Human, 13-15; *Eye Neoplasms--Genetics--GE; *Retinoblastoma--Genetics--GE; Child, Preschool; Chromosome Fragile Sites; Chromosomes, Human, 1-3; Chromosomes, Human, 16-18; Karyotyping; Metaphase

8/5/26 (Item 1 from file: 351)
DIALOG(R)File 351:DERWENT WPI

009158942 WPI Acc No: 92-286380/35
XRAM Acc No: C92-127359
Chromosome-specific staining - to detect genetic re-arrangements associated with cancers
Patent Assignee: (REGC) UNIV CALIFORNIA
Author (Inventor): GRAY J W; KALLIONIEMI A; KALLIONIEMI O; PINKEL D; SAKAMOTO M
Number of Patents: 004
Number of Countries: 018
Patent Family:

CC	Number	Kind	Date	Week	
EP	500290	A2	920826	9235	(Basic)
CA	2060267	A	920823	9246	
EP	500290	A3	930303	9349	
JP	6038798	A	940215	9411	

Priority Data (CC No Date): US 659974 (910222); US 670242 (910315) Applications (CC,No,Date): JP 9272635 (920221); EP 92301266 (920217); CA 2060267 (920128); EP 92301266 (920217)
Language: English
EP and/or WO Cited Patents: No-SR.Pub; 4.Jnl.Ref; CA 2021489 X; EP 293266 A; EP 430402 D; FR 2627192 A; WO 9005789 X
Designated States (Regional): AT; BE; CH; DE; DK; ES; FR; GB; GR; IT; LI; LU; MC; NL; PT; SE Abstract (Basic): EP 500290 A

A method of staining targetted chromosomal material based upon nucleic acid (NA) sequence employs high complexity NA probes, where the staining pattern produced is indicative of the presence or absence of one or more genetic rearrangements involving chromosomes 3, 13, 17 and/or a tumour suppressor gene.

Also claimed are: (1) highly complex Na probes for the detection of one or more genetic rearrangements associated with the retinoblastoma tumour suppressor gene, chromosome 2 and/or chromosome 17 in humans; (2) a method of detecting the genetic rearrangements of (I) comprising: (a) hybridising the probes of (I) to targetted chromosomal material; (b) observing and/or measuring the proximity of and/or other characteristics of the signals from the probes; and (c) determining whether one or more genetic rearrangements have occurred; and (3) a chromosome specific staining reagent that provides staining patterns indicative of the genetic rearrangements of (1), produced by the process of: (a) isolating chromosome-specific DNA from chromosomes 3, 13 and/or 17; (b) amplifying pieces of isolated chromosome-specific DNA; (c) disabling the hybridising capacity of and/or removing shared repetitive sequences contained in the amplified pieces of the isolated DNA to form a collection of NA fragments which hybridise predominantly to targetted chromosomal DNA or

chromasomes 3, 13 and 17; and (d) labelling the NA fragments to form a heterogeneous mixt. of NA fragments.

USE/ADVANTAGE - The method allows staining of the targetted chromosomal material whether it is in metaphase or interphase. Cells from clinical specimens may be stained e.g. those suspected of being cancerous. Particularly associated with genetic rearrangements, notably deletions in the p arm of chromosome 3 are small cell lung cancer, renal cell cancer, uterine and/or ovarian cancers. Cancers associated with deletion in the q arm of chromosome 13 are retinoblastomas, osteosarcomas, small cell lung cancer and breast cance Dwg.0/9

File Segment: CPI
Derwent Class: B04; D16;
Int Pat Class: C12N-015/10; C12Q-001/68; G01N-033/48
Manual Codes (CPI/A-N): B04-B04A1; B11-C07B1; B12-K04A3; D05-H09; D05-H12 Chemical Fragment Codes (M1):
01 M423 M750 M903 N102 P831 Q233 V753
Chemical Fragment Codes (M6):
02 M903 P633 P831 Q233 R515 R521 R614 R627 R639

8/5/27 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

139812 DBA Accession No.: 92-12304 PATENT
Chromosome-specific staining to detect genetic rearrangement - DNA probe for detection of cancer associated with chromosome-3, chromosome-13 and/or chromosome-17 rearrangement
PATENT ASSIGNEE: Univ.Calif. 1992
PATENT NUMBER: EP 500290 PATENT DATE: 920826 WPI ACCESSION NO.: 92-286380 (9235)
PRIORITY APPLIC. NO.: US 670242 APPLIC. DATE: 910315
NATIONAL APPLIC. NO.: EP 92301266 APPLIC. DATE: 920217
LANGUAGE: English
ABSTRACT: A new method for staining targeted chromosomal material (metaphase and/or interphase chromosomes) is based on nucleic acid sequence employing high complexity nucleic acid probes where the staining pattern produced is indicative of the presence or absence of at least 1 genetic rearrangement (preferably translocations, inversions, insertions, aneuploidy, amplifications or deletions) involving chromosome-3, chromosome-13 and/or chromosome-17 and causing cancer (preferably retinoblastoma, osteosarcoma, lung cancer, mamma carcinoma, renal cell cancer (RCC), ovary cancer and/or uterus cancer). The rearrangement is a 13q deletion within the retinoblastoma tumor suppressor gene causing retinoblastoma, osteosarcoma, small cell lung cancer (SCLC) and mamma carcinoma. The high complexity nucleic acid probe is preferably a reference probe, especially a centromere-specific alpha-satellite probe. Alternatively, the rearrangement is a 3p deletion associated with SCLC, RCC, uterus cancer and ovary

cancer. The DNA probes and methods of tumor diagnosis involving polymerase chain reaction amplification are also new. (35pp)
DESCRIPTORS: DNA probe appl. human chromosome-3, chromosome-13, chromosome-17 rearrangement det., retinoblastoma, osteosarcoma, lung cancer, mamma carcinoma, renal cell cancer, ovary cancer, uterus cancer diagnosis mammal tumor
SECTION: Microbiology-Genetics; Pharmaceuticals-Other (A1,D5)

Set	Items	Description
S1	5297	AU=GRAY, J? OR AU=GRAY J?
S2	32084	AU=PINKEL? OR AU=KALLIONIEMI? OR AU=SAKAMOTO?
S3	37151	S1 OR S2
S4	711	CHROMOSOM?(3N)3 AND CHROMOSOM?(3N)17
S5	19	RETINOBLASTOMA? AND S4
S6	14	S4 AND S3
S7	31	S5 OR S6
S8	27	RD (unique items)

APS
08/478,387
9.2.96
CHK'D, 1M

1. 5,547,838, Aug. 20, 1996, Method for the rapid and ultra-sensitive detection of leukemic cells; Paul E. Nisson, et al., 435/6, 91.2, 91.51; 536/24.31 [IMAGE AVAILABLE]

US PAT NO: 5,547,838 [IMAGE AVAILABLE] L5: 1 of 28

ABSTRACT:
An improved method is disclosed for diagnosing the presence of a **chromosomal** translocation characteristic of acute myelogenous leukemia. **Nucleic** acid molecules that may be used in this improved method are described.

2. 5,538,869, Jul. 23, 1996, In-situ hybridization probes for identification and banding of specific **human** **chromosomes** and regions; Michael J. Siciliano, et al., 435/91.2, 6; 536/24.3, 24.31 [IMAGE AVAILABLE]

US PAT NO: 5,538,869 [IMAGE AVAILABLE] L5: 2 of 28

ABSTRACT:
The invention relates to novel primer sets useful in preparing **DNA** probes specific for any **chromosome** or part of a **chromosome**, particularly **human** **chromosomes**. The **DNA** probes so produced may be used to paint individual **chromosomes** or portions of **chromosomes** in metephase cell spreads and in interphase nuclei. When used to paint **chromosomes** in metephase spreads, R-bands are readily detectable. The method is sensitive and has been shown to paint R-bands on **chromosomes** pieces having as few as several hundred kilobases.

3. 5,538,846, Jul. 23, 1996, BCL-1 locus **nucleic** acid probes and assay methods; Timothy C. Meeker, 435/6, 91.2; 536/24.33; 935/8, 77, 78 [IMAGE AVAILABLE]

US PAT NO: 5,538,846 [IMAGE AVAILABLE] L5: 3 of 28

ABSTRACT:
Nucleic acid probes substantially complementary to **nucleic** acid sequences within the bcl-1 locus are disclosed as well as polymerase chain reaction (PCR) primers substantially complementary to **nucleic** acid sequences within that locus that are useful in detecting t(11;14)(q13;q32) translocations associated with hematopoietic cancers. Further, the bcl-1 locus probes are useful in detecting bcl-1 amplifications found in about twenty percent of solid tumors, particularly in squamous cell and mammary carcinomas.
Diagnostic/prognostic methods for cancer are disclosed, as well as cancer research methods using the bcl-1 probes of this invention.

4. 5,534,418, Jul. 9, 1996, Controlled expression of recombinant proteins; Roland M. Evans, et al., 435/69.7, 172.3; 536/24.1 [IMAGE AVAILABLE]

US PAT NO: 5,534,418 [IMAGE AVAILABLE] L5: 4 of 28

ABSTRACT:
The present invention provides methods for the controlled production of recombinant proteins in cells. Cells employed in the invention method contain a gene encoding the desired recombinant protein, with transcription of the gene maintained under the control of a transcriptional control element which is activated by a ligand/receptor complex. The ligand/receptor complex is formed when a ligand (which is a hormone or/and analog thereof) is complexed with a receptor (which is a hormone receptor or functional analog thereof which has the transcription activating properties of the receptor). Receptor is produced by the expression of non-endogenous **DNA** which is also present in the cells used for production of recombinant protein.

5. 5,527,676, Jun. 18, 1996, Detection of loss of the wild-type P53 gene and kits therefor; Bert Vogelstein, et al., 435/6, 69.1, 810; 436/63, 501; 514/44; 536/23.1, 24.1, 24.31, 24.32, 24.33; 935/77, 78 [IMAGE AVAILABLE]

US PAT NO: 5,527,676 [IMAGE AVAILABLE] L5: 5 of 28

ABSTRACT:
Methods and kits are provided for assessing mutations and/or loss of the p53 gene in **human** tumors. Both deletion mutations and point mutations in p53 are observed in the same **human** tumor cells and these mutations are clonal within the cells of the tumor. Loss of wild-type p53 genes is responsible for neoplastic progression.

6. 5,518,899, May 21, 1996, Preparation of **human** myelomonocyte interferon-gamma; Masashi Kurimoto, et al., 435/70.5; 424/85.5; 435/240.2; 530/351 [IMAGE AVAILABLE]

US PAT NO: 5,518,899 [IMAGE AVAILABLE] L5: 6 of 28

ABSTRACT:
The present invention relates to a novel **human** interferon-gamma derived from an established **human** myelomonocyte, a process to prepare said interferon-gamma, and its use. The **human** myelomonocyte interferon-gamma has a novel polypeptide and carbohydrate chain structure, and it is effective in preventing and treating viral diseases, malignant tumors and immunopathies alone or in combination with other lymphokine and/or chemotherapeutic. The **human** myelomonocyte interferon-gamma may be produced by culturing an established **human** myelomonocyte on a culture medium in vitro. Alternatively, an established **human** myelomonocyte is implanted in a non-**human** warm-blooded animal or in a diffusion chamber placed inside or outside the body of the animal, and then allowed to proliferate while receiving nutrient body fluid from the animal. The **human** myelomonocyte may be contacted with an inducer during propagation.

7. 5,508,164, Apr. 16, 1996, Isolation of biological materials using magnetic particles; Albert P. Kausch, et al., 435/6, 7.2, 82 [IMAGE AVAILABLE]

US PAT NO: 5,508,164 [IMAGE AVAILABLE] L5: 7 of 28

ABSTRACT:
A method for the isolation and sorting of biological materials has been developed. Biological material includes **chromosomes**, segments of **chromosomes**, cell organelles, or other minute cellular components. The biological material is separated from the cellular milieu, if necessary, and anchored to a support. Example of a support are glass coverslips, glass or polymer beads. The anchoring is by means of a reversible cross-linking system. The supported biological material is then labelled with compositions capable of binding to said material, and with magnetic particles. Examples of the binding material include **nucleic** acid probes and antibodies. An example of the antibodies would be those directed to histones. Other labels, for example, fluoresceinbiotin-avidin may be used. The material may be released from the support and sorted by a magnetic force. This method is an alternative to flow cytometry and presents numerous advantages in terms of time, resolution, purity, and preservation of the structure of the biological material during isolation and separation.

8. 5,472,842, Dec. 5, 1995, Detection of amplified or deleted **chromosomal** regions; Trond Stokke, et al., 435/6; 536/24.31 [IMAGE AVAILABLE]

US PAT NO: 5,472,842 [IMAGE AVAILABLE] L5: 8 of 28

ABSTRACT:
The present invention relates to in situ hybridization methods for the identification of new **chromosomal** abnormalities associated with various diseases. In particular, it provides probes which are specific to a region of amplification in **chromosome** 20.

9. 5,468,629, Nov. 21, 1995, Method of promoting in vitro homologous recombination transfection in mammalian cells using the RecA protein; Cornelia Calhoun, 435/172.3, 240.2 [IMAGE AVAILABLE]

US PAT NO: 5,468,629 [IMAGE AVAILABLE] L5: 9 of 28

ABSTRACT:
A rapid method for transfection of a cell under physiological conditions suitable to the survival and growth of the cell is disclosed. According to the method, a stable nucleoprotein complex is provided. The nucleoprotein complex comprises a single-stranded **DNA** sequence in stable combination with RecA protein molecules. Cells to be transformed are cultured in a

physiologically suitable medium to which the nucleoprotein complex has been added. As the cells grow and undergo mitosis, the nucleoprotein complex is taken up within some of the cells and becomes integrated into the genome. The method accomplishes transfection without resort to infectious vectors or permeabilization or other manipulation of the cell membrane. According to another object of the invention, a diagnostics method is provided. A directly detectable reporter label or an indirectly detectable ligand is bound to the nucleoprotein complex to provide a **DNA** probe which then is taken up into the cell and integrates into the cell's genome. Upon appropriate treatment the detectable reporter label can be observed or the ligand can be reacted with a suitable detectable reporter molecule to allow visualization and thereby confirm whether the compliment of the **DNA** sequence in the probe is substantially present in the genome of the cell.

10. 5,468,617, Nov. 21, 1995, Steroid/thyroid hormone receptor-related gene, which is inappropriately expressed in **human** heptocellular carcinoma, and which is a retinoic acid receptor; Hughes Blaudin De The, et al., 435/7.8, 7.1; 436/63, 501; 530/350 [IMAGE AVAILABLE]

US PAT NO: 5,468,617 [IMAGE AVAILABLE] L5: 10 of 28

ABSTRACT:

A previously isolated hepatitis B virus (HBV) integration in a 147 bp cellular **DNA** fragment linked to hepatocellular carcinoma (HCC) was used as a probe to clone the corresponding complementary **DNA** from a **human** liver cDNA library. Nucleotide sequence analysis revealed that the overall structure of the cellular gene, which has been named hap, is similar to that of the **DNA**-binding hormone receptors. Six out of seven hepatoma and hepatoma-derived cell-lines express a 2.5 kb hap mRNA species which is undetectable in normal adult and fetal livers, but present in all non-hepatic tissues analyzed. Low stringency hybridization experiments revealed the existence of hap related genes in the **human** genome. The cloned **DNA** sequence is useful in the preparation of pure hap protein and as a probe in the detection and isolation of complementary **DNA** and **RNA** sequences. The hap protein is a retinoic acid (RA) receptor identified as RAR-.beta.. The RAR-.beta. gene is transcriptionally up-regulated by retinoic acid (RA) and its promoter region may contain a RARE (retinoic acid responsive element).

11. 5,449,616, Sep. 12, 1995, **Nucleic** acid encoding dystrophin-associated protein; Kevin P. Campbell, et al., 435/240.2, 252.3, 320.1; 530/350; 536/23.5 [IMAGE AVAILABLE]

US PAT NO: 5,449,616 [IMAGE AVAILABLE] L5: 11 of 28

ABSTRACT:

Disclosed are **nucleic** acid sequences encoding components of the dystrophin-glycoprotein complex. The components include dystroglycan, the 50 kDa protein component and the 59 kDa protein component. Also disclosed are compositions and methods which

relate to the disclosed sequences.
12. 5,449,604, Sep. 12, 1995, **Chromosome** 14 and familial Alzheimers disease genetic markers and assays; Gerard D. Schellenberg, et al., 435/6, 91.2 [IMAGE AVAILABLE]

US PAT NO: 5,449,604 [IMAGE AVAILABLE] L5: 12 of 28
ABSTRACT:
Method for isolating a **DNA** segment indicative of an Alzheimer's disease trait in a family population, wherein said family population consists essentially of a plurality of blood relatives of an individual having a **chromosome** 14 Alzheimer's disease trait, by: preparing a test sample of immobilized separated genomic **DNA** fragments from a plurality of the blood relatives, contacting each of the test samples with a test oligonucleotide under conditions permitting hybridization of complementary single stranded **DNA** molecules, wherein the test oligonucleotide is complementary with at least a portion of a genetic marker located between band q11.2 and band q32.1 in **chromosome** 14, identifying a plurality of hybridized molecules so formed as alleles of the genetic marker in the family population, identifying one of the genetic marker alleles as indicative of the Alzheimer's disease trait in the family population by either determining by pedigree analysis a segregation value for each of the genetic markers alleles and the Alzheimer's disease trait, and selecting an indicative genetic marker allele that co-segregates with the Alzheimer's disease trait in the family population, or measuring genetic linkage between each of the genetic marker alleles and the Alzheimer's disease trait, and selecting a genetic marker allele as indicative of the Alzheimer's disease trait in the family population if the selected genetic marker allele has a maximal LOD score of at least 3 at a recombination fraction of about 0.0 to about 0.1 for genetic linkage with the Alzheimer's disease trait in the family population, and isolating a **chromosome** 14 **DNA** segment containing the indicative genetic marker allele.

13. 5,438,126, Aug. 1, 1995, **Human** thyroid hormone receptor **DNA**; Leslie J. DeGroot, et al., 536/23.5; 435/69.1, 69.4, 240.2, 320.1; 935/11, 23 [IMAGE AVAILABLE]

US PAT NO: 5,438,126 [IMAGE AVAILABLE] L5: 13 of 28
ABSTRACT:
The **DNA** encoding **human** thyroid hormone receptor protein alpha-1 (hTR -1). This polynucleotide can be used as a probe to detect the presence of a third form of **human** thyroid hormone receptor protein.
14. 5,427,932, Jun. 27, 1995, Repeat sequence **chromosome** specific **nucleic** acid probes and methods of preparing and using; Heinz-Ulrich G. Weier, et al., 435/91.2, 6, 810; 436/501; 536/22.1, 23.1, 24.3, 24.31, 24.33; 935/78, 88 [IMAGE AVAILABLE]

US PAT NO: 5,427,932 [IMAGE AVAILABLE] L5: 14 of

28
ABSTRACT:
A primer directed **DNA** amplification method to isolate efficiently **chromosome**-specific repeated **DNA** wherein degenerate oligonucleotide primers are used is disclosed. The probes produced are a heterogeneous mixture that can be used with blocking **DNA** as a **chromosome**-specific staining reagent, and/or the elements of the mixture can be screened for high specificity, size and/or high degree of repetition among other parameters. The degenerate primers are sets of primers that vary in sequence but are substantially complementary to highly repeated **nucleic** acid sequences, preferably clustered within the template **DNA**, for example, pericentromeric alpha satellite repeat sequences. The template **DNA** is preferably **chromosome**-specific. Exemplary primers ard probes are disclosed. The probes of this invention can be used to determine the number of **chromosomes** of a specific type in metaphase spreads, in germ line and/or somatic cell interphase nuclei, micronuclei and/or in tissue sections. Also provided is a method to select arbitrarily repeat sequence probes that can be screened for **chromosome**-specificity.

15. 5,427,910, Jun. 27, 1995, Method of cytogenetic analysis; Louis A. Kamentsky, et al., 435/6, 810; 436/63, 501; 935/77, 78 [IMAGE AVAILABLE]
US PAT NO: 5,427,910 [IMAGE AVAILABLE] L5: 15 of 28
ABSTRACT:
A method of characterizing the **chromosomes** in a sample of cells by fixing the cell sample on a substrate, contacting the cell sample with a **nucleic** acid probe having a detectable label under conditions that allow the probe to hybridize preferentially to a **chromosome** in the cells to form a hybridized complex, optically detecting each labeled complex in the sample, defining a predetermined number of neighboring labeled complexes as a group, generating a distance parameter based on the distance between the position of a group and the position of the next neighboring labeled complex, and comparing the distance parameter for each group to a standard distance value to characterize the **chromosomes** in the cells of the sample.

16. 5,413,907, May 9, 1995, Diagnosis for malignant hyperthermia; Ronald G. Worton, et al., 435/6; 536/23.5, 24.31 [IMAGE AVAILABLE]
US PAT NO: 5,413,907 [IMAGE AVAILABLE] L5: 16 of 28
ABSTRACT:

A method for isolating a cDNA specific for the **human** ryanodine receptor is disclosed. The gene is associated with malignant hyperthermia, a hypermetabolic syndrome triggered primarily by inhalation anesthetics. The cDNA can be cloned and expressed in a recombinant plasmid or phage. The cDNA, or

fragments thereof, is used as diagnostic probes for individuals at risk for malignant hyperthermia using restriction fragment length polymorphism analysis. The cDNA is that sequenced in FIG. 2 of this specification.

17. 5,411,859, May 2, 1995, Genetic identification employing **DNA** probes of variable number tandem repeat loci; Raymond L. White, et al., 435/6, 91.2, 172.3, 320.1; 436/63; 536/22.1, 23.1, 24.1, 24.3, 24.31, 24.33; 935/77, 78 [IMAGE AVAILABLE]

US PAT NO: 5,411,859 [IMAGE AVAILABLE] L5: 17 of 28

ABSTRACT:

The present invention is related to the identification of cloned **DNA** sequences that reveal individual multiallele loci. The loci are used in the process of the present invention to provide convenient and accurate genetic identification. A large number of clones that recognize VNTR loci have been isolated from a cosmid library and characterized.

18. 5,376,530, Dec. 27, 1994, Steroid/thyroid hormone receptor-related gene, which is inappropriately expressed in **human** heptocellular carcinoma, and which is a retinoic acid receptor; Hughes B. De The, et al., 435/6, 69.1, 172.3; 530/326, 327, 350, 828; 536/23.1, 24.31 [IMAGE AVAILABLE]

US PAT NO: 5,376,530 [IMAGE AVAILABLE] L5: 18 of 28

ABSTRACT:

A previously isolated hepatitis B virus (HBV) integration in a 147 bp cellular **DNA** fragment linked to hepatocellular carcinoma (HCC) was used as a probe to clone the corresponding complementary **DNA** from a **human** liver cDNA library. Nucleotide sequence analysis revealed that the overall structure of the cellular gene, which has been named hap, is similar to that of the **DNA**-binding hormone receptors. Six out of seven hepatoma and hepatoma-derived cell-lines express a 2.5 kb hap mRNA species which is undetectable in normal adult and fetal livers, but present in all non-hepatic tissues analyzed. Low stringency hybridization experiments revealed the existence of hap related genes in the **human** genome. The cloned **DNA** sequence is useful in the preparation of pure hap protein and as a probe in the detection and isolation of complementary **DNA** and **RNA** sequences. The hap protein is a retinoic acid (RA) receptor identified as RAR-.beta.. The RAR-.beta. gene is transcriptionally up-regulated by retinoic acid (RA) and its promoter region may contain a RARE (retinoic acid responsive element).

19. 5,362,490, Nov. 8, 1994, **Human** myelomonocyte interferon-gamma, and process for preparation and use thereof; Masashi Kurimoto, et al., 424/85.5, 85.1, 85.2, 85.6, 85.7; 435/70.5; 530/351, 413 [IMAGE AVAILABLE]

US PAT NO: 5,362,490 [IMAGE AVAILABLE] L5: 19 of 28

ABSTRACT:
The present invention relates to a novel **human** interferon-gamma derived from an established **human** myelomonocyte, a process to prepare said interferon-gamma, and its use. The **human** myelomonocyte interferon-gamma has a novel polypeptide and carbohydrate chain structure, and it is effective in preventing and treating viral diseases, malignant tumors and immunopathies alone or in combination with other lymphokine and/or chemotherapeutic.

20. 5,317,090, May 31, 1994, Steroid/thyroid hormone receptor-related gene, which is inappropriately expressed in **human** hepatocellular carcinoma, and which is a retinoic acid receptor; Hughes Blaudin De The, et al., 530/387.1, 387.9, 388.1, 388.22, 391.1 [IMAGE AVAILABLE]

US PAT NO: 5,317,090 [IMAGE AVAILABLE] L5: 20 of 28

ABSTRACT:
A previously isolated hepatitis B virus (HBV) integration in a 147 bp cellular **DNA** fragment linked to hepatocellular carcinoma (HCC) was used as a probe to clone the corresponding complementary **DNA** from a **human** liver cDNA library. Nucleotide sequence analysis revealed that the overall structure of the cellular gene, which has been named hap, is similar to that of the **DNA**-binding hormone receptors. Six out of seven hepatoma and hepatoma-derived cell-lines express a 2.5 kb hap mRNA species which is undetectable in normal adult and fetal livers, but present in all non-hepatic tissues analyzed. Low stringency hybridization experiments revealed the existence of hap related genes in the **human** genome. The cloned **DNA** sequence is useful in the preparation of pure hap protein and as a probe in the detection and isolation of complementary **DNA** and **RNA** sequences. The hap protein is a retinoic acid (RA) receptor identified as RAR-.beta.. The RAR-.beta. gene is transcriptionally up-regulated by retinoic acid (RA) and its promoter region may contain a RARE (retinoic acid responsive element).

21. 5,312,732, May 17, 1994, Hormone receptor compositions and methods; Ronald M. Evans, et al., 435/69.1, 240.2, 252.3, 320.1; 536/23.5 [IMAGE AVAILABLE]

US PAT NO: 5,312,732 [IMAGE AVAILABLE] L5: 21 of 28

ABSTRACT:
The present invention provides substantially pure **DNA**'s comprised of sequences which encode proteins having the hormone-binding and/or transcription-activating characteristics of a glucocorticoid receptor, a mineralocorticoid receptor, or a thyroid hormone receptor. The invention also provides various plasmids containing receptor sequences which exemplify the **DNA**'s of the invention. The invention further provides receptor proteins, including modified functional forms thereof, expressed from the **DNA**'s (or mRNA's) of the invention. In addition to the novel receptor **DNA**, **RNA** and protein

compositions, the present invention involves a bioassay for determining the functionality of a receptor protein. By using our bioassay system we have discovered that a necessary and sufficient condition for activation of transcription of a gene (G), whose transcription is activated by hormones complexed with receptors, is the presence of the hormone and its receptor in the cell (C) where (G) is located. As a result of that discovery we have also invented new methods for producing desired proteins in genetically engineered cells. Two of these methods are methods of the present invention. The first is a method for inducing transcription of a gene whose transcription is activated by hormones complexed with the receptors. The second is a method for engineering a cell and increasing and controlling production of a protein encoded by a gene whose transcription is activated by hormones complexed with receptor proteins.

22. 5,298,429, Mar. 29, 1994, Bioassay for identifying ligands for steroid hormone receptors; Ronald M. Evans, et al., 436/501; 435/69.1, 172.3, 252.3, 320.1 [IMAGE AVAILABLE]

US PAT NO: 5,298,429 [IMAGE AVAILABLE] L5: 22 of 28

ABSTRACT:
Bioassays are disclosed which are useful for determining whether a compound is a hormone receptor agonist (i.e., is capable of promoting the transcription-activation activities of such receptors) or a hormone receptor antagonist (i.e., is capable of blocking the
transcription-activation activities of such receptors). The invention bioassay is conducted by culturing test cells in the presence of at least one compound whose ability to function as a ligand for said receptor protein (or functional engineered or modified forms thereof) is sought to be determined. Alternatively, test cells are cultured in medium containing increasing concentrations of at least one compound whose ability to inhibit the transcription activation activity of hormone receptor agonists is sought to be determined, and a fixed concentration of at least one agonist for the receptor protein. Test cells employed in the practice of the present invention contain non-endogenous **DNA** which expresses hormone receptor (or functional modified forms thereof) and a **DNA** sequence encoding a hormone response element operatively linked to a reporter gene. The cultured cells are monitored for evidence of transcription of the reporter gene as a function of the concentration of test compound in the culture medium. The variation in transcription levels of the reporter gene as a function of concentration of test compound indicates the ability of test compound to promote or inhibit activation of transcription.

23. 5,242,795, Sep. 7, 1993, TCL-5 gene rearrangement involved in T-cell leukemia and melanoma; Carle M. Croco, 435/6, 91.2; 536/23.1; 935/77, 78 [IMAGE AVAILABLE]

US PAT NO: 5,242,795 [IMAGE AVAILABLE] L5: 23 of 28
ABSTRACT:
A gene is described which is involved in the neoplastic process of a number of different cancers. The gene, termed TCL-5, is located at **chromosome** 1, band 32, adjacent to the **chromosome** junction formed during a t(1;14)(p32;q11) translocation. Probes and primers for detecting TCL-5 rearrangements are provided, as well as methods for detecting abnormalities in TCL-5 expression.

24. 5,149,628, Sep. 22, 1992, Methods for detecting bcl-3 gene in **human** leukemias; Carlo M. Croce, 435/6, 5, 7.23, 172.3; 436/501, 538, 813; 536/24.31; 935/2, 19 [IMAGE AVAILABLE]

US PAT NO: 5,149,628 [IMAGE AVAILABLE] L5: 24 of 28
ABSTRACT:
Bcl-3 gene sequences can be used to monitor the success of chemotherapy and to detect minimal residual disease in patients having hematopoietic malignancies. Bcl-3 sequences can also be used to identify new cellular oncogenes involved in hematopoietic cell malignancies by taking advantage of their adjacency to the bcl-3 promoter. Particular **nucleic** acid probes and primers are also provided by the disclosure.

25. 5,098,823, Mar. 24, 1992, **Chromosome**-specific **nucleic** acid probe for familial Polyposis coli; Walter F. Bodmer, et al., 435/6, 252.3, 320.1; 436/501; 536/24.31; 935/77, 78 [IMAGE AVAILABLE]
US PAT NO: 5,098,823 [IMAGE AVAILABLE] L5: 25 of 28
ABSTRACT:

A **nucleic** acid fragment capable of selectively hybridizing with the **human** **chromosome** 5 at the **chromosomal** region 5q20-q23 is disclosed. Also disclosed are probes which include the fragment bearing a detectable level as well as processes for presymptomatic screening for FAP and processes for the pathological classification of colonic tumors and precancerous polyps.

26. 5,071,773, Dec. 10, 1991, Hormone receptor-related bioassays; Ronald M. Evans, et al., 436/501; 435/69.7, 172.3 [IMAGE AVAILABLE]
US PAT NO: 5,071,773 [IMAGE AVAILABLE] L5: 26 of 28
ABSTRACT:
The present invention discloses two hormone receptor-related bioassays. The first bioassay is useful for determining whether a protein suspected of being a hormone receptor has transcription-activating properties of a hormone receptor. The second bioassay is useful for evaluating whether compounds are functional ligands for receptor proteins. According to the first

bioassay, cells that contain non-endogenous **DNA** which expresses a protein suspected of being a hormone receptor and which contain a **DNA** sequence encoding an operative hormone responsive
promoter/enhancer element linked to an operative reporter gene, are cultured, the culturing being conducted in a culture medium containing a known hormone, or an analog thereof. The cultured cells are then monitored for induction of the product of the reporter gene as an indication of functional transcription-activating binding between the hormone or hormone analog and the protein suspected of being a hormone receptor. According to the second bioassay, cells that contain non-endogenous **DNA** which expresses hormone receptor or a functional engineered or modified form thereof, and which also contain a **DNA** sequence encoding an operative hormone responsive promoter/enhancer element linked to an operative reporter gene, are cultured, the culturing being conducted in culture medium containing at least one compound whose ability to functionally bind the receptor protein is sought to be determined. The cultured cells are then monitored for induction of the product of the report gene as an indicator of functional binding between the compound and the receptor.

27. 4,963,663, Oct. 16, 1990, Genetic identification employing **DNA** probes of variable number tandem repeat loci; Raymond L. White, et al., 536/24.31; 435/6; 436/63; 536/24.3; 935/10, 19, 29, 73, 78 [IMAGE AVAILABLE]

US PAT NO: 4,963,663 [IMAGE AVAILABLE] L5: 27 of 28
ABSTRACT:

The present invention is related to the identification of cloned **DNA** sequences that reveal individual multiallele loci. The loci are used in the process of the present invention to provide convenient and accurate genetic identification. A large number of clones that recognize VNTR loci have been isolated from a cosmid library and characterized.

28. 4,728,614, Mar. 1, 1988, Mutant **human** T cell line producing immunosuppressive factor and method for obtaining such mutants; Catherine Y. Lau, 435/240.2, 70.4, 172.1, 948; 530/351 [IMAGE AVAILABLE]

US PAT NO: 4,728,614 [IMAGE AVAILABLE] L5: 28 of 28
ABSTRACT:

A stable mutant **human** T cell line is disclosed which secretes a high titer suppressor inducer factor. This suppressor inducer factor in turn induces production of a T cell suppressor factor which suppressed mitogen-induced T cell proliferation at high dilution. Also disclosed is a general method for mutating lymphoblastoid cell lines to yield mutants secreting enhanced levels of lymphokines.

(FILE 'USPAT' ENTERED AT 14:06:27 ON 02 SEP 96)

L1 4159 S GRAY?/IN OR PINKEL?/IN OR KALLIONIEMI?/IN OR SAKAMOTO?/I N
L2 19072 S DNA OR RNA OR NUCLEIC
L3 2765 S HUMAN AND CHROMOSOM? AND L2
L4 34 S CHROMOSOM?(3A)3 AND CHROMOSOM?(3A)17
L5 28 S L3 AND L4
L6 2 S L5 AND L1